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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/202,984	03/19/1999	ARMIN PETER CZERNILOFSKY	0652.1830000	3631

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EXAMINER

CHUNDURU, SURYAPRABHA

ART UNIT	PAPER NUMBER
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1637

DATE MAILED: 04/19/2004

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary

Application No.

09/202,984

Applicant(s)

CZERNILOFSKY ET AL.

Examiner

Suryaprabha Chunduru

Art Unit

1637

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
 - If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
 - If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
 - Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 11 January 2004.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 61-120 is/are pending in the application.
- 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 61-120 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some * c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
 2. ☐ Certified copies of the priority documents have been received in Application No. _____.
 3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- | | |
|--|---|
| 1) <input checked="" type="checkbox"/> Notice of References Cited (PTO-892) | 4) <input type="checkbox"/> Interview Summary (PTO-413)
Paper No(s)/Mail Date. _____ |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948) | 5) <input type="checkbox"/> Notice of Informal Patent Application (PTO-152) |
| 3) <input type="checkbox"/> Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08)
Paper No(s)/Mail Date _____ | 6) <input type="checkbox"/> Other: _____ |

DETAILED ACTION

1. Acknowledgement is made for the request to establish continued prosecution application (RCE) filed on January 11, 2004. The request for RCE is accepted and is established with the status of the application as follows:
 - a. the filing date of this RCE is established as 3/19/1999;
 - b. Claims 1-60 are cancelled. New claims 61-120 are added.
2. Applicants' response to the earlier office action filed along with RCE is considered and has been entered.

Response to Arguments

3. Applicants' response to the office action is fully considered and found persuasive.
4. With reference to the rejections made in the previous office action under 103(a), Applicants' arguments and the amendment are fully considered and the rejections are withdrawn herein in view of canceling the claims 28-60 in the instant amendment.

New Rejections

Claim Rejections - 35 USC § 102

5. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless –

(e) the invention was described in (1) an application for patent, published under section 122(b), by another filed in the United States before the invention by the applicant for patent or (2) a patent granted on an application for patent by another filed in the United States before the invention by the applicant for patent, except that an international application filed under the treaty defined in section 351(a) shall have the effects for purposes of this subsection of an application filed in the United States only if the international application designated the United States and was published under Article 21(2) of such treaty in the English language.

A. Claims 61-64, 66-70, 77-83, 87-93, 95-99, 106-112, 116-120 are rejected under 35

U.S.C. 102(e) as being anticipated by Vande Woude et al. (USPN. 5,645,988).

Vande Woude et al. teach parallel screening method of claims 61-62, 90-91, of determining the pharmacological effect of a substance (anti-cancer drug) on the activity of different biological target molecules contained in test cells (cancer cells) of same type, comprising

(a) applying or contacting a drug (appropriate concentration (see column 12, lines 45-60)) to test cells (cancer cells) derived from the same type of biological material (see column 11, lines 36-57), wherein the cancer cells differ in the presence of a particular target molecule (oncogene, proto oncogene, tumor suppressor genes) (see column 5, lines 39-46, column 11, lines 24-30, lines 59-67, column 12, lines 1-4, column 7, lines 1-21);

(b) measuring the effect of the substance on the biological activities (cell growth, tumor formation and the phenotypes of transformed cells (see column 7, lines 22-29) of said different target molecules using detection system (see column 5, lines 46-47, column 11, lines 30-32);

(c) comparing the effect of said test substance on the biological activities of said different target molecules, wherein said biological activities are selected from the group of metabolically coupled signal transduction (cell cycle pathway genes) (see column 5, lines 47-50, column 7, lines 30-60, column 11, lines 32-35).

With regard to claims 63-64, 66, 92-93, 95, Vande Woude et al. teach that said different target molecules include Ras (K-ras, N-ras, H-ras), vmos-raf (see column 12, lines 1-41, column 7, lines 30-50, column 1-2, table 1);

With regard to claims 61, 67-70, 96-99, Vande Woude et al. teach said biological activity is receptor-coupled signal transduction including tyrosine kinase serine/threonine kinases growth

factor receptors such as EGF (see column 6, lines 20-29, column 7, lines 30-49, column 1-2, table 1);

With regard to claims 77-80, 106-109, Vande Woude et al. teach that the biological activity is a pathological effect including proliferation, or apoptosis (see column 7, lines 22-50, column 11, lines 36-58);

With regard to claims 87-89, 116-117, Vande Woude et al. teach that said test cells are mammalian cells comprising human cancer cells having same genotype (see column 11, lines 36-59, column 34, lines 49-51);

With regard to claims 90-91, Vande Woude et al. teach that said method is determines effect of a test substance on the same biological target molecule (mos) contained in test cells of different or same types but of with a different state of differentiation or activation (column 7, lines 30-49, wherein mos is differentially expressed in different stages of cell division);

With regard to claims 81-82, 110-111, Vande Woude et al. teach that said test cells are transformed with DNA operably encoding said different target molecules which include receptors (see column 13, lines 7-32, column 1-2, table.1);

With regard to claims 83, 112, Vande Woude et al. teach said detection system comprises proliferation assay, an apoptosis assay (see column 38, lines 59-67, column 39, lines 1-18, column 42, lines 54-67, column 43, lines 1-39, column 45, lines 3-17);

With regard to claims 118-120, Vande Woude et al. also teach that said test cells are from different or same types or same type with different states of differentiation and include tumor and normal cells (see column 7, lines 30-49). Thus the disclosure of Vande Woude et al. meets the limitations in the instant claims.

B. Claims 61-62, 67-73, 77-79, 90-91, 96-102, 106-108 are rejected under 35 U.S.C. 102(e) as being anticipated by Tang et al. (USPN. 5,710,173).

Tang et al. teach parallel screening method (96-well microtiter plates) of claims 61-62, 90-91, of determining the pharmacological effect of a substance (anti-cancer drug) on the activity of different biological target molecules contained in test cells of same type, comprising

(a) applying or contacting a drug (appropriate concentration (see column 19, lines 47-49), to test cells derived from the same type of biological material, wherein the test cells differ in the presence of a particular target molecule (tyrosine kinases) (see column 10, lines 58-62);

(b) measuring the effect of the substance on the biological activities (cell growth), (see column 10, lines 62-63) of said different target molecules using detection system (see column 11, lines 4-26);

(c) comparing the effect of said test substance on the biological activities of said different target molecules, wherein said biological activities are selected from the group of metabolically coupled signal transduction (tyrosine kinase pathway) (see column 11, lines 20-26, column 2, lines 25-30, column 4, lines 16-26).

With regard to claims 61, 67-71, 96-100, Tang et al. teach said biological activity is receptor-coupled signal transduction including tyrosine kinase, growth factor receptors such as EGF, HGF, VEGF (see column 5, lines 7-23, column 7, lines 49-65, column 9, lines 60-67, column 10, lines 1-29);

With regard to claims 72-73, 101-103, Tang et al. teach that said different target molecules include EGF, HGF, HER2, KDR (see column 4, lines 56-62, column 5, lines 7-23, column 7, lines 49-65, column 9, lines 60-67, column 10, lines 1-29);

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With regard to claims 77-79, 106-108, Tang et al. teach that the biological activity is a pathological effect including proliferation, and differentiation (see column 4, lines 16-26). Thus the disclosure of Tang et al. meets the limitations in the instant claims.

Claim Rejections - 35 USC § 103

6. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

A. Claims 74-76, 84-85, 103-105, 113-114 are rejected under 35 U.S.C. 103(a) as being unpatentable over Vande Woude et al. (USPN. 5,645,988) in view of Czernilofsky et al. (USPN. 5,854,004).

Vande Woude et al. teach parallel screening method of claims 60-61, 90-91, of determining the pharmacological effect of a substance (anti-cancer drug) on the activity of different biological target molecules contained in test cells (cancer cells) of same type, comprising

(a) applying or contacting a drug (appropriate concentration (see column 12, lines 45-60)) to test cells (cancer cells) derived from the same type of biological material (see column 11, lines 36-57), wherein the cancer cells differ in the presence of a particular target molecule (oncogene, proto oncogene, tumor suppressor genes) (see column 5, lines 39-46, column 11, lines 24-30, lines 59-67, column 12, lines 1-4, column 7, lines 1-21);

(b) measuring the effect of the substance on the biological activities (cell growth, tumor formation and the phenotypes of transformed cells (see column 7, lines 22-29) of said different target molecules using detection system (see column 5, lines 46-47, column 11, lines 30-32);

(c) comparing the effect of said test substance on the biological activities of said different target molecules, wherein said biological activities are selected from the group of metabolically coupled signal transduction (cell cycle pathway genes) (see column 5, lines 47-50, column 7, lines 30-60, column 11, lines 32-35). Vande Woude et al. also teach that said different target molecules include Ras (K-ras, N-ras, H-ras), vmos-raf (see column 12, lines 1-41, column 7, lines 30-50, column 1-2, table 1); Vande Woude et al. teach said biological activity is receptor-coupled signal transduction including tyrosine kinase serine/threonine kinases growth factor receptors such as EGF (see column 6, lines 20-29, column 7, lines 30-49, column 1-2, table 1).

However, Vande Woude et al. did not teach other target molecules which include other than tyrosine kinases. Serine/ threonine kinases such as G-protein coupled receptors, neurokinin receptors (neurokinin-1 neurokinin-2), or serotonin receptors (5HT₂) and detection system comprising reporter system selected from the group consisting of luciferase, alkaline phosphatase, β -glucuronidase, chloramphenicol-acetyltransferase.

Czernilofsky et al. teach a parallel screening method of determining pharmacological effect of a substance on different receptor-coupled signal pathway effector targets, wherein the method comprises transforming test cells with particular receptor gene sequences under the control of TRE-regulated reporter gene expression system to effect the signaling of a particular signal transduction pathway and measuring the effect of gene expression signal in the presence of a test substance and comparing the effect with control cells (see column 8, lines 49-67, column 9, lines 1-47). Czernilofsky et al. also disclose that (i) the target receptors include receptors coupled with phospholipase C-signal transduction pathway such as serotonin receptors (5HT_{1c}, 5HT₂), human neurokinin receptors (NK1, NK2, NK3), FGF receptors, PDGF receptors (see column 14, lines 8-35); (ii) suitable reporter genes include luciferase, alkaline phosphatase, β -glucuronidase, and chloramphenicol-acetyltransferase (see column 10, lines 20-56).

Therefore, it would have been obvious to one of ordinary skill in the art at the time the invention was made to combine the method of determining the effect of a substance on the biological activities on target molecules as taught by Vande Woude et al. with the method of detecting effect of a substance on different target molecules linked to a reporter gene system as taught by Czernilofsky et al. to achieve an expected advantage of developing a versatile and sensitive method of detecting the effect of a substance on biological activities of a wide range of target molecules with the aid of a sensitive reporter detection system because Czernilofsky et al. taught the advantage of monitoring the effect of a test substance on multistage receptor dependent intracellular signal transduction pathway to discover pharmacologically active substances for different types of indications and with a choice of specific receptor or receptor subtypes, to distinguish with great specificity between key mechanisms in different cell systems.

Further the expression of the receptor indicated by the expression of the reporter gene after the receptor has been activated by the binding to a ligand aids in cloning of receptors which are biochemically characterized (see column 17, lines 29-51).

Therefore an ordinary practitioner would have been motivated to combine the method of detecting the effect of a substance on the biological activities of different target molecules as taught by Vande Woude et al. with the different receptor target molecules linked to a reporter system as taught by Czernilofsky et al. to achieve in developing an improved and sensitive method for detecting the effect of a substance on various target molecules that activate a signal transduction pathway(s) thereby characterizing said activated target molecules and detecting the corresponding signal transduction pathway mediated by said receptor molecules. Additionally it would aid in cloning the drug or substance activated receptor molecules to develop an improved method for designing an appropriate treatment regime for a particular pathological condition in which said receptor mediated signal transduction pathway is activated.

B. Claims 86 and 115 are rejected under 35 U.S.C. 103(a) as being unpatentable over Vande Woude et al. (USPN. 5,645,988) in view of Czernilofsky et al. (USPN. 5,854,004). as applied to claims 74-76, 84-85, 103-105, 113-114 above, and further in view of Chalfie et al. (USPN.5,491,084).

Vande Woude et al. teach parallel screening method of determining the pharmacological effect of a substance (anti-cancer drug) on the activity of different biological target molecules contained in test cells (cancer cells) of same type, comprising

(a) applying or contacting a drug (appropriate concentration (see column 12, lines 45-60)) to test cells (cancer cells) derived from the same type of biological material (see column 11, lines

36-57), wherein the cancer cells differ in the presence of a particular target molecule (oncogene, proto oncogene, tumor suppressor genes) (see column 5, lines 39-46, column 11, lines 24-30, lines 59-67, column 12, lines 1-4, column 7, lines 1-21);

(b) measuring the effect of the substance on the biological activities (cell growth, tumor formation and the phenotypes of transformed cells (see column 7, lines 22-29) of said different target molecules using detection system (see column 5, lines 46-47, column 11, lines 30-32);

(c) comparing the effect of said test substance on the biological activities of said different target molecules, wherein said biological activities are selected from the group of metabolically coupled signal transduction (cell cycle pathway genes) (see column 5, lines 47-50, column 7, lines 30-60, column 11, lines 32-35). Vande Woude et al. also teach that said different target molecules include Ras (K-ras, N-ras, H-ras), vmos-raf (see column 12, lines 1-41, column 7, lines 30-50, column 1-2, table 1); Vande Woude et al. teach said biological activity is receptor-coupled signal transduction including tyrosine kinase serine/threonine kinases growth factor receptors such as EGF (see column 6, lines 20-29, column 7, lines 30-49, column 1-2, table 1).

Vande Woude et al. did not teach other target molecules which include other than tyrosine kinases. Serine/ threonine kinases such as G-protein coupled receptors, neurokinin receptors (neurokinin-1 neurokinin-2), or serotonin receptors (5HT₂) and detection system comprising reporter system selected from the group consisting of luciferase, alkaline phosphatase, β -glucuronidase, chloramphenicol-acetyltransferase.

Czernilofsky et al. teach a parallel screening method of determining pharmacological effect of a substance on different receptor-coupled signal pathway effector targets, wherein the method comprises transforming test cells with particular receptor gene sequences under the

control of TRE-regulated reporter gene expression system to effect the signaling of a particular signal transduction pathway and measuring the effect of gene expression signal in the presence of a test substance and comparing the effect with control cells (see column 8, lines 49-67, column 9, lines 1-47). Czernilofsky et al. also disclose that (i) the target receptors include receptors coupled with phospholipase C-signal transduction pathway such as serotonin receptors (5HT_{1c}, 5HT₂), human neurokinin receptors (NK1, NK2, NK3), FGF receptors, PDGF receptors (see column 14, lines 8-35); (ii) suitable reporter genes include luciferase, alkaline phosphatase, β -glucuronidase, and chloramphenicol-acetyltransferase (see column 10, lines 20-56).

Neither Vande Woude et al. nor Czernilofsky et al. teach green fluorescent protein as a reporter gene.

Chalfie et al. teach a method for cells expressing a biological activity (gene expression) of a particular target molecule, wherein the regulatory sequences of a target molecule are linked to a reporter fluorescent protein which fluoresces when said target is expressed within the cells (see column 1, lines 38-52). Chalfie et al. also teach that said reporter fluorescent protein is a gene encoding a green fluorescent protein (column 1, lines 38-41).

Therefore, it would have been obvious to one of ordinary skill in the art at the time the invention was made to combine the method of determining the effect of a substance on the biological activities on target molecules as taught by Vande Woude et al. with the method of detecting effect of a substance on different target molecules linked to a reporter gene system as taught by Czernilofsky et al. to achieve an expected advantage of developing a versatile and sensitive method of detecting the effect of a substance on biological activities of a wide range of target molecules with the aid of a sensitive reporter detection system because Czernilofsky et al.

taught the advantage of monitoring the effect of a test substance on multistage receptor dependent intracellular signal transduction pathway to discover pharmacologically active substances for different types of indications and with a choice of specific receptor or receptor subtypes, to distinguish with great specificity between key mechanisms in different cell systems. Further the expression of the receptor indicated by the expression of the reporter gene after the receptor has been activated by the binding to a ligand aids in cloning of receptors which are biochemically characterized (see column 17, lines 29-51).

Therefore an ordinary practitioner would have been motivated to combine the method of detecting the effect of a substance on the biological activities of different target molecules as taught by Vande Woude et al. with the different receptor target molecules linked to a reporter system as taught by Czernilofsky et al. to achieve in developing an improved and sensitive method for detecting the effect of a substance on various target molecules that activate a signal transduction pathway(s) thereby characterizing said activated target molecules and detecting the corresponding signal transduction pathway mediated by said receptor molecules. Additionally it would aid in cloning the drug or substance activated receptor molecules to develop an improved method for designing an appropriate treatment regime for a particular pathological condition in which said receptor mediated signal transduction pathway is activated.

Further it would have been obvious to combine the reporter gene mediated detection of the effect of a substance on the biological activities with the method of selecting or localizing a biological activity within the cells using the reporter gene encoding a green fluorescent protein as taught by Chalfie et al. to achieve an enhanced sensitivity in determining the effect of a substance on the biological activity or activities because Chalfie et al. taught that the biological

activity of a particular target molecule in response to an external stimulus can be monitored within the cells containing the target by the expression of green fluorescence protein linked to said target and the cells expressing the GFP can be easily selected and sorted by a fluorescent-activated sorter (see column 4, lines 3-12). Therefore an ordinary practitioner would have motivated to combine the reporter gene mediated detection method of determining the effect of a substance on the biological activity with the method of selecting or localizing a biological activity within the cells using the reporter gene encoding a green fluorescent protein as taught by Chalfie et al. to enhance the detection of the biological activity of a target molecule within the cells, so as to detect and sort the cells expressing the target molecules without lysing the cells.

C. Claims 65, and 94 are rejected under 35 U.S.C. 103(a) as being unpatentable over Vande Woude et al. (USPN. 5,645,988) in view of Reed et al. (USPN. 5,686,595).

Vande Woude et al. teach parallel screening method of determining the pharmacological effect of a substance (anti-cancer drug) on the activity of different biological target molecules contained in test cells (cancer cells) of same type, comprising

(a) applying or contacting a drug (appropriate concentration (see column 12, lines 45-60)) to test cells (cancer cells) derived from the same type of biological material (see column 11, lines 36-57), wherein the cancer cells differ in the presence of a particular target molecule (oncogene, proto oncogene, tumor suppressor genes) (see column 5, lines 39-46, column 11, lines 24-30, lines 59-67, column 12, lines 1-4, column 7, lines 1-21);

(b) measuring the effect of the substance on the biological activities (cell growth, tumor formation and the phenotypes of transformed cells (see column 7, lines 22-29) of said different target molecules using detection system (see column 5, lines 46-47, column 11, lines 30-32);

(c) comparing the effect of said test substance on the biological activities of said different target molecules, wherein said biological activities are selected from the group of metabolically coupled signal transduction (cell cycle pathway genes) (see column 5, lines 47-50, column 7, lines 30-60, column 11, lines 32-35). Vande Woude et al. also teach that said different target molecules include Ras (K-ras, N-ras, H-ras), vmos-raf (see column 12, lines 1-41, column 7, lines 30-50, column 1-2, table 1); Vande Woude et al. teach said biological activity is receptor-coupled signal transduction including tyrosine kinase serine/threonine kinases growth factor receptors such as EGF (see column 6, lines 20-29, column 7, lines 30-49, column 1-2, table 1). However, Vande Woude et al. did not teach other target molecules which include Bcl-2.

Reed et al. teach a method for screening agents that inhibit binding of Bcl-2 related polypeptide with Bcl-2 target molecule (see column 8, lines 15-27), Reed et al. also teach regulation of Bcl-2 expression by modulating the binding properties of Bcl-2 with Bcl-2-related agents in cancer cells would reduce the level of free Bcl-2 in a cell and modulate the susceptibility of a cell to apoptosis (see column 24-49).

Therefore an ordinary practitioner would have been motivated to combine the method of detecting the effect of a substance on the biological activity of different target molecules as taught by Vande Woude et al. with the different target molecules as taught by Reed et al. to achieve in developing an improved method for detecting the effect of a substance on a wide range of target molecules that affect not only cell proliferation activity but also apoptosis activity because Reed et al. taught the advantage of monitoring Bcl-2 expression in apoptosis which enables to discover pharmacologically active substances that modulate the expression of Bcl-2 and apoptosis (see column 11, lines 45-49). An ordinary practitioner would have been

motivated to combine the method of detecting the effect of a substance on the biological activity of different target molecules of Vande Woude et al. with inclusion of Bcl-2 target molecule which would result in improving the method of detecting the effect of a substance on not only the biological activity related to cell proliferation but also the effect of a substance on the biological activity related to apoptosis which would also aid in identifying drug targets related to apoptosis.

Conclusion

No claims are allowable.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Suryaprabha Chunduru whose telephone number is 571-272-0783. The examiner can normally be reached on 8.30A.M. - 4.30P.M, Mon - Friday.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Gary Benzion can be reached on 571-272-0782. The fax phone numbers for the organization where this application or proceeding is assigned are 703-872-9306 for regular communications and - for After Final communications.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the receptionist whose telephone number is 703-308-0196.

⁹⁸
Suryaprabha Chunduru
April 8, 2004


JEHANNE SITTON
PRIMARY EXAMINER

4/9/04